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Learned Helplessness in the Perspective of the Depressive Disorders: Conceptual and Definitional Issues

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This article examines three issues that are important in extending the learned helplessness model to clinical depressive disorders. First, the nature of the heterogeneity of clinical manifestations of depression is examined within several clinical frameworks, and the role that learned helplessness may play in each is discussed. Second, the problems in constructing models for clinical populations are explored through the examination of several parallels between learned helplessness and clinical depression put forth by Seligman. Third, problems involved in defining and identifying depressed college student subjects in analogue research are discussed. Integral to the latter issue is an evaluation of the assumption that depressed college student subjects differ from clinical depressives only quantitatively but not qualitatively. The article concludes with suggestions for future research aimed at extending the learned helplessness model to other clinical problem areas.

Research in the area of learned helplessness has proliferated in the past 5 years with impressive theoretical and empirical developments. The investigation of learned helplessness notions has illuminated the behavioral deficits noted in escape conditioning in many species, including man, found subsequent to exposure to uncontrollable circumstances (Seligman, 1975). Moreover, learned helplessness provides a unifying theme for the investigation of diverse areas, such as biochemical alterations subsequent to acute and chronic inescapable shock, human laboratory research on the parameters of stress, and field research on stressful life events.

One of the more exciting extensions of recent helplessness studies is the analogue research, where learned helplessness is viewed as a model of naturally occurring depression in man. This area of inquiry is in a state of rapid development (witness this issue), and already investigators are discussing further extensions to clinical depressive populations. For instance, Klein and Seligman (1976) recently wrote, "So, generalizing our results to the psychopathology of depression seems reasonable, and preliminary results of replications of some of our findings with inpatients are encouraging" (p. 25). Moreover, interest in testing the efficacy of therapies for depression within a learned helplessness framework is growing. Klein and Seligman (1976) recently indicated that their findings "suggest a basic design for testing in the laboratory the effectiveness of any therapy for depression" (p. 24), and they proceeded to discuss this possibility.

In view of these developments, it now appears to be a crucial time to examine the learned helplessness model within the perspective of clinical depressive disorders. The remainder of this article, then, focuses on three significant issues that will require consideration in further extensions of the model to clinical populations.

The first issue concerns the heterogeneity of clinical manifestations inherent in the depressive disorders. The nature of this heterogeneity is examined, albeit briefly in view of space limitations, within several clinical frameworks, and the role that learned helplessness may play in each is discussed. The second issue relates to the process of con-

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structing models for clinical depressive groups. We examine several parallels between learned helplessness and clinical depression put forth by Seligman (1975) and discuss the problems and complexities that will need to be addressed in future extensions of the model. The third issue concerns the problems involved in defining and identifying depressed college student subjects in analogue research. Integral to this discussion is an evaluation of the assumption that depressed college student subjects differ from clinical depressives only quantitatively but not qualitatively. The article concludes with suggestions for future research aimed at extending the learned helplessness model to other clinical problem areas.

Before proceeding, we wish to emphasize that our comments are put forth in the spirit of promoting the extension of the model to clinical populations and not solely as criticisms of learned helplessness notions or their proponents. Their work has been provocative and, therefore, merits serious theoretical and empirical challenging to establish the boundaries of its applicability. It is toward this goal that our comments are directed; we hope they are received accordingly.

Learned Helplessness in the Perspective of the Depressive Disorders

As students of psychopathology are too well aware, the clinical manifestations of the depressive disorders are enormously heterogeneous with respect to signs and symptoms, levels of severity, and clinical course. This fact, as well as the equal diversity in theoretical notions concerning the etiology of depressions, has generated heated debates over which the nosological framework best conceptualizes the clinical phenomena. Fortunately, the last 15 years have witnessed a healthy growth in empirical work in this area, and we may now have just tipped the scales in favor of data as opposed to debate. If one point is clear from this work, it is that the clinical issues are exceedingly more complex than originally thought, and it is the nature of these complexities that we wish to introduce into discussions of learned helplessness as a model of clinical depression.

Recent research on the depressive disorders has attempted to delimit clinical heterogeneity through the delineation of subgroups based on differences in symptoms, clinical course characteristics, genetic and biological variables, and treatment response factors. The important question for researchers is to which subgroups the learned helplessness model is applicable, and how the concept of helplessness interrelates with other suspected etiologic factors associated with the various subtypes of depressive disorders. Hence, in this section, we address these questions within several clinical frameworks. First, we focus on the concept of reactive depression, since learned helplessness has been viewed as a model of this form of depression. Subsequently, we examine several forms of depression previously neglected in helplessness theorizing, that of situational, minor, and major depressive disorders, and comment briefly on their relation to the learned helplessness model of depression.

Concept of Reactive Depression

Over the past 65 years, numerous issues in the depressive disorders related to etiology, symptom characteristics, and severity have coalesced into the concept of endogenous versus reactive depression. Currently, many investigators use this distinction interchangeably with endogenous–neurotic and psychotic–neurotic distinctions, even though there are conceptual differences in the meaning of endogenous–reactive as opposed to psychotic–neurotic depression (Beck, 1967; Kendell, 1976; Mendels & Cochrane, 1968). In keeping with Fowles and Gersh (in press), we prefer the use of endogenous–neurotic because this is the most often used combination and because neurotic premorbid personalities and neurotic symptoms are more clearly related to the "reactive" groups than is the presence of a precipitating stressor (Fowles & Gersh, in press; Kendell, 1976; Paykel, 1971, 1972a, 1972b).

Concurrent with theoretical debates concerning the endogenous–reactive/neurotic distinction, researchers have attempted to define the differential symptom complexes, thera-

peutic response patterns, and outcomes associated with endogenous and neurotic depressions. Concerning the issue of whether differences exist between the extreme representations of these groups, there is generally good agreement that the two groups have different features. However, the issue of whether these two groups should be viewed as related along one or more dimensions or whether they form two or more distinct categories (apparently, the Seligman position; see quote below) remains unsettled, although most researchers favor a dimensional approach (Fowles & Gersh, in press; Kendell, 1976). Regardless of one's position, it is clear that obvious boundaries between endogenous and neurotic groups, if they exist at all, are not yet evident (Kendell, 1976).

Another problem in this area concerns the fact that in spite of the general agreement that endogenous and neurotic groups differ in their extreme forms, the groups differ with respect to the clarity of their features. For instance, as Kendell (1976) and Fowles and Gersh (in press) have concluded, studies have consistently produced a cluster or factor identifiable with endogenous depression but not with reactive/neurotic depression. Also relatively clear is that the *presence or absence of a precipitating event is much less important in differentiating the two groups* or in predicting response to electroconvulsive shock than are other features, such as reactivity to environmental changes *during the depression* or neurotic features (Fowles & Gersh, in press). Indeed, several authors have concluded that the etiologic role of stressful environmental changes is always one of degree rather than a simple question of presence or absence (Depue, in press-b; Kendell, 1976; Paykel, 1974, in press).

For our purposes, more important than the semantic confusion associated with this distinction is the research that illustrates the tremendous degree of heterogeneity inherent in the reactive group of patients (Fowles & Gersh, in press; Gersh & Fowles, in press; Kendell, 1976). Indeed, it has become impossible to speak meaningfully of reactive depression as a unified entity or type or to speak of this group confidently within the framework of a single etiologic factor. Surprisingly, in suggesting that learned helplessness best serves as a model for reactive depression, Seligman's statements reflect little of the confusion in this area and imply definite positions on issues of etiology:

> The most useful and best-confirmed typology of depression is the endogenous–reactive dichotomy. The reactive depressions are by far the most common, and the kind familiar to us all. Roughly 75 percent of all depressions are reactions to some external event, such as the death of a child. Reactive depressions do not cycle regularly in time, are not usually responsive to physical therapies like drugs and electroconvulsive shock (ECS), are not genetically predisposed, and are usually somewhat milder in their symptoms than endogenous depression. . . . Endogenous depressions . . . are not triggered by any external event; they just sweep over the sufferer. (Seligman, 1975, p. 78)[1]

What remains most problematic for the learned helplessness model is that few studies have produced a factor with any confidence identifiable with reactive/neurotic depression (Kendell, 1976). Indeed, neurotic depression is enormously heterogeneous and has been viewed variously as being mixed and diluted with other syndromes (depression representing a common symptom; Mendels & Cochrane, 1968), as a diffuse entity encompassing some of the ways in which individuals utilize defense mechanisms to cope with neuroticism and concurrent environmental stress (Kiloh, Andrews, Neilson, & Bianchi, 1972), as a residue left behind by the removal of endogenous features (Foulds, 1973), and as a nonspecific

[1] This statement assumes confirmation on many issues with respect to the endogenous–reactive distinction: (a) A typology rather than dimensionality is the best model for conceiving the endogenous–reactive distinction; (b) the typology is based on etiologic grounds (reactive depression is a reaction to external events, whereas endogenous depression is not triggered by events but develops as a result of some autonomous biologic process); (c) the reactive depressions are usually unresponsive to physical therapies; and (d) the reactive depressions are not genetically predisposed. Several recent reviews offer detailed arguments on many of these points, and the reader is referred to their excellent discussions (Becker, 1974; Fowles & Gersh, in press; Gersh & Fowles, in press; Kendell, 1976). Suffice it here to say that most of these assumptions are as yet unsettled, whereas some are definitely not supported (e.g., the etiologic role of stress) or lack evidence bearing on support.

Table 1
Features of Paykel's (1971, 1972a, 1972b) Four-Group Typology of Depression

Psychotic type
1. oldest
2. ideas of guilt and hopelessness of delusional intensity
3. highest scores on typically endogenous symptoms (distinct quality of depression, anorexia, weight loss, retardation, and delayed insomnia)
4. low scores on a recent life events questionnaire
5. low scores on the Maudsley Personality Inventory Neuroticism scale[a]
Anxious type
1. second oldest
2. highest scores on psychic and somatic anxiety, depersonalization, obsessional symptoms, fatigue
3. moderately depressed
4. highest scores on suicidal tendencies, number of previous episodes of depression, and the Maudsley Personality Inventory Neuroticism scale
5. *low* scores on a recent life events inventory and reactivity to environment during the depression
Hostile type
1. younger
2. particularly high scores on hostility and self-pity in relation to scores on other symptoms
3. moderate depression
4. relatively high Maudsley Personality Inventory Neuroticism scores
Young depressives with personality disorder type
1. youngest
2. mildly depressed
3. high Maudsley Personality Inventory Neuroticism, recent life events inventory, and reactivity scores
4. tendency to receive clinical diagnoses of personality disorders
5. high incidence of hysterical personality traits

[a] From Eysenck (1964).

stress reaction of a depressive type that is not limited to neurotic patients (Pilowsky, Levine, & Boulton, 1969). Moreover, Akiskal (in press) recently reported on an operational study on the diagnostic usage of "neurotic depression" and found that in addition to situational depression, it referred to neurotic syndromes with secondary depression (close to Mendels & Cochrane's, 1968, conception), characterological depression, Bipolar 2 disease (where depressive and hypomanic episodes occur, but hospitalizations result only from depressions), and to nonpsychotic forms of primary unipolar depression.

In light of such heterogeneity, research is needed to clarify the nature of neurotic depressions. Paykel (1971, 1972a, 1972b) has provided to date the clearest evidence that subtypes of neurotic depression need to be distinguished. This study is important because of the inclusion of a large group of outpatients, the use of cluster analysis to assign patients to mutually exclusive diagnostic categories, and the collection of additional evidence supporting the validity of the subtypes that were generated. Table 1 presents the features of the four groups that resulted from these efforts.

Inspection of Table 1 indicates that this four-group classification is somewhat consistent with—though more differentiated than—a psychotic–neurotic distinction. The psychotic group clearly corresponds to a psychotic or endogenous group. Paykel (1971, 1972a) suggests that the other three groups may all be regarded as neurotic in that they

display some neurotic symptoms and have poor premorbid adjustment, as evidenced by high scores on the Maudsley Personality Inventory (MPI) Neuroticism scale (Eysenck, 1964). Hospital diagnoses provided some support for this interpretation in that most patients in the psychotic group were diagnosed psychotic, and most of the other three groups were labeled psychoneurotic depressive reaction. However, the lack of clear boundaries is evident in that 27% of the psychotic group were diagnosed psychoneurotic depressive reaction, 21% of the anxious depressives were labeled psychotic, and sizable fractions of the hostile (15.9%) and young depressive (20.8%) groups received diagnoses of personality disorders. Another complexity was that the anxious depressive group did not fit the stereotype of reactive depression because of low scores on reactivity during depression and on precipitating events. This group also showed a poor response to placebo and imipramine but a good response to phenothiazines and/or diazepam. These findings suggest an important subdivision within the neurotic depressions in which anxiety depressives are separated from other groups. Indeed, intensive study is now aimed at whether a boundary exists between anxiety depressives and anxiety neurotics, but thus far no consensus exists (Gersh & Fowles, in press).[2]

As this brief review indicates, the notion that learned helplessness may provide a model for research on reactive/neurotic depressions needs revision in view of current formulations concerning problems inherent with this classificatory scheme. Which subdivisions are most applicable to the learned helplessness paradigm is, as previously stated by Klein and Seligman (1976), an empirical question. Yet, it is a question that should be explored within a clinical framework that is sensitive to the heterogeneity inherent in the reactive/neurotic depressions.

Although the application of a learned helplessness model may need revision with respect to the reactive/neurotic depressions classification, the utility of the model for other prevalent forms of depression may be worthy of exploration.

Situational Depression

One of the most troublesome issues raised above for the learned helplessness model is that there is not strong support or, at best, very inconsistent support for the notion that neurotic depressions arise solely as a reaction to stressful life events. And yet, the quote from Seligman (1975) above and many of his case illustrations place much weight on this etiologic framework. Indeed, most of his discussions possess the features of what Akiskal (in press) refers to as "situational" depression, which he uses in preference to "reactive" depression in order to counter any implications that other types of depressions are unrelated to life events. Akiskal views situational depression as continuous with the transient states of depressed mood that we all experience with the following additional features: It lasts longer (several weeks to several months); typically, a specific life event precedes the depressive state; the person is painfully preoccupied with the event, which forms the central theme of the depressive condition; the depressed mood lasts as long as the aversive situation persists; and full recovery is expected when the life situation is reversed or changes for the better.

The prevalence of such depressions is quite high in general population surveys (Comstock & Helsing, 1976), but their relation to clinical depression is obscure. For example, one of the most common forms of situational depressions is grief, defined as an understandable response to the death of a loved person or object. Such depressive responses are ordinarily self-limited and rarely lead to seeking psychiatric help or hospitalization (Akiskal, in press; Clayton, Desmarais, & Winokur, 1968). Furthermore, Parkes (1972) has argued that preoccupation with the image of the lost object (sometimes to the point of hallucinosis), anxiety, agitation, somatic symptoms, and difficulty falling

[2] It should be noted that Paykel's four-group classification gains further support as a research generating scheme in that other researchers have found factors or profiles with some similarity to anxious depression, hostile depression, and young depressives with personality disorder (Fowles & Gersh, in press).

Table 2
General Features of Minor Depressive Disorders

Episodic disorder
1. nonpsychotic episodes of illness lasting at least 1 week
2. a relatively sustained mood of depression without the full depressive syndrome that characterizes major depressive disorder[a]
3. episodes preceded and followed by normal mood of at least 2-months duration with relatively clear-cut onset and offset
4. two or more of specified symptoms[a]
5. impairment in social functioning with family, at home, at school, or at work; taking medication or seeking or being referred for clinical help
Chronic and intermittent disorder
1. for at least past 2 years, bothered by depressed mood much of the time with intermittent periods of normal mood lasting from a few days to a few weeks
2. two or more of specified symptoms[a]
3. impairment in social functioning with family, at home, at school, or at work; taking medication or seeking or being referred for clinical help

[a] See Spitzer et al.'s (1975) *Research Diagnostic Criteria Manual.*

asleep—rather than depression per se—constitute the symptomatologic hallmarks of such separation responses in grief (as well as in other responses to loss). Moreover, as Akiskal (in press) notes, it may be unwise to generalize from studies of grief to other forms of depression elicited by separation. For instance, the available data indicate that the depressive syndrome that occurs in divorcing individuals is often the cause rather than the result of divorce (Briscoe & Smith, 1975).

Whatever the nature and prevalence of situational depressions, they rarely present for psychiatric treatment, and generalization of such cases to clinically depressed populations is not yet warranted (Akiskal, in press). This is not to say that it is always easy to distinguish clinically between a transient depressive response to life events and depressive illness. Nor is it to say that learned helplessness is not an adequate model for such responses. Indeed, the model may well contribute to an understanding of why grief carries an increased risk for developing depresssive illness (the risk being 15% in children [Spitz, 1946] and not more than 5% in adults [Parkes, 1972]). It may be that one factor that increases the vulnerability of the individuals at higher risk concerns expectations of inability to control stress, as suggested by Garber, Miller, and Seaman (in press) in a recent extension of the learned helplessness model. These issues present a challenge for future research. The point is that we must be clear about the nature of the populations to which we are referring and precise about the parameters used in constructing models (a point we shall return to later).

Minor Depressive Disorders

In the last 10 years, there has been a renewed interest in diagnosis and classification. As part of this trend, researchers have attempted to refine the diagnostic procedure through the use of standardized structured interview formats and through the development of research diagnostic criteria (RDC). The most elaborate RDC system was developed by Spitzer, Endicott, and Robins (1975). This system defines and offers diagnostic criteria for two forms of minor depressive disorders: episodic as well as chronic and intermittent. The full criteria may be found in the RDC manual,[3] so we have provided only their general characteristics, which may be viewed in Table 2.

It is our belief that these forms of depressive disorders have been neglected as poten-

[3] The *Research Diagnostic Criteria Manual* may be purchased for $1 from Robert L. Spitzer, Biometrics Research, New York State Psychiatric Institute, 722 West 168th Street, New York, New York 10032.

tially fruitful areas for the extension of the learned helplessness model. The potential importance of these disorders was illustrated by Garber et al.'s (in press) recent extensions of the model. They argue that individuals who develop a generalized expectation of inability to control future outcomes are particularly vulnerable to the development of learned helplessness and depression. The exciting implications of this formulation are that these individuals may develop personality predispositions characterized by the expectancy of lack of control over life stressors and, hence, may exhibit exaggerated and/or more persistent feelings of helplessness and depression in the face of numerous life circumstances (which may be stressful for either objective or subjective reasons).

One other factor suggests the importance of the minor depressive disorders. They are differentiated from major depressive disorders and also from very mild forms of manic depressive disorder (cyclothymic personality, in which there exists a pattern of cycling from depression to better than normal mood), from generalized anxiety disorder (in which there is a clear preponderance of anxious mood), and from labile personality (in which the depressed mood rarely lasts more than a few hours or days at a time, and the mood changes are abrupt in onset). Although major depressive disorder may subsequently occur, it does not have to, and thus it may be that these minor disorders represent distinct problem areas with etiologies differing from other depressive disorders. Only further research will shed light on this possibility.

Major Depressive Disorders

The notion that learned helplessness is a model of depression is equally important in consideration of the major depressive disorders. However, as was the case in reactive/neurotic depressions, the major depressions are enormously heterogeneous. Numerous researchers have suggested that in terms of research-generating potential, the major depressive disorders (see the Spitzer et al., 1975, manual for diagnostic criteria) may be most effectively divided into bipolar and unipolar groups (Depue & Monroe, in press-a, in press-b; Kendell, 1976). It is no longer possible to provide a simple definition for these two groups due to the fact that the clinical manifestations within each are so variable. The variability results from the application of a genetic taxonomy to depressive disorders (Depue & Monroe, in press-a). This system employs a familial definition of polarity, where family history for affective disorder serves as one index of classification. Thus, along with phenotypic (observable) behavior, the family history of the individual (the presence or absence of manic or hypomanic disorder in first-degree relatives) becomes the determining criterion on which polarity is based; phenotypic expression *by itself* is, therefore, not the determining factor and may, in fact, be variable within a polar group.

When such a system is applied to individuals requiring treatment, several unipolar and bipolar subgroups may be formed on the basis of severity of illness of manic or depressive symptoms as well as on family history for affective disorder. The various unipolar and bipolar subgroups and their definitions suggested in the recent literature may be seen in Table 3. It should be emphasized that it is not presently known whether all of these subtypes will be valid or even useful distinctions. The value of the system has been "measured" in terms of its research generating potential.

Future investigations aimed at exploring the contribution of learned helplessness to the onset of major depressive disorders need to differentiate between unipolar and bipolar disorders. As may be seen in Table 4, Bipolar 1 and Unipolar 1 groups exhibit important differences in behavioral symptomatology, clinical course, genetic factors, and pharmacological response (see Depue & Monroe, in press-a, for a thorough review). Theoretically, they may differ also with respect to the impact of psychosocial stressors (Depue & Monroe, in press-b). This may be important for the moderating effects of the learned helplessness notion concerning the expectancy of lack of control over life stressors (Garber et al., in press).

The etiologic role of learned helplessness in the major depressive disorders will most likely need to be redefined. In most discussions of

Table 3
Subtypes of Unipolar and Bipolar Disorders

Type	Definition
	Bipolar
Bipolar 1	personal history of both depressive and manic episodes, both usually requiring hospitalizations
Unipolar mania	at least one hospitalization of mania, but no episodes of depression; family history of bipolar disorder
Bipolar 2	hospitalizations for depressive episodes, but no manic or hypomanic symptoms leading to hospitalization; family history of bipolar disorder
Bipolar 3[a]	at least one hospitalization for depression; no personal history of mania or hypomania, but family history of bipolar disorder
Bipolar other	treated on an outpatient basis for depressive and hypomanic symptoms, but never hospitalized
	Unipolar[b]
Unipolar 1[c]	at least one hospitalization for depression; no personal nor family history of mania or hypomania
Neurotic	relation to Unipolar 1 unsettled
Unipolar other	sought help or been treated for depressions only, but not hospitalized; no family history of bipolar disorder

[a] Fieve (1975) places this group within unipolar disorder (referred to as Unipolar 2). However, genetic researchers separate the former group out of unipolar samples in their studies, and it is more in keeping with a genetic taxonomy to refer to these cases as bipolar.
[b] The unipolar group represents all or most of the remaining recurrent depressions lumped together in a single heterogeneous group, including the traditional involutional, neurotic, and psychotic reaction types.
[c] Winokur, Cadoret, Baker, and Dorzab (1975) have suggested that the Unipolar 1 group may be divided into depressive spectrum disease and pure depressive disease. See their discussion for details.

the model, learned helplessness is easily construed as both a necessary and *sufficient* condition for depression. The notion of a single etiologic factor in these disorders or in most psychopathologic and psychosomatic disorders (Depue, in press-b) does not do justice to the array of implicated or supported variables now under consideration, not the least of which are genetic factors (Depue & Monroe, in press-a). Indeed, the necessity of considering numerous biological, psychosocial, and developmental variables in the onset of these disorders has been emphasized recently in several integrative reviews (Akiskal, in press; Depue & Evans, in press). Akiskal (in press) has specifically discussed learned helplessness as one potential contributor within an elaborate, integrated model of depressive disorders. His framework may serve to provide a perspective within which further extensions of the learned helplessness model to major depressive disorders may proceed.

Establishing Analogies between Learned Helplessness and Clinical Depression

Learned helplessness is viewed as a model of naturally occurring (reactive) depression in man. The process of testing this model involves the drawing of analogies between variables found in learned helplessness studies and variables observed in depression. The process of constructing models requires careful specification of the variables in each analogy, and often rules of correspondence are made explicit. The analogue studies of learned helplessness and depression, where depressed college students have been employed, have been most carefully conceived and executed with respect to the modeling process. In extensions of this modeling process, where clinical depressive populations are the focus, researchers will need to draw analogies with extreme precision. The reasons for this should now be obvious: There are many different forms and variations within forms of depressive disorders, each with varying symptom patterns,

Table 4
Characteristics of Bipolar 1 and Unipolar 1 Depressed Patients

Area	Bipolar 1	Unipolar 1
Behavior		
Psychomotor	retardation, minimal pacing	usually active pacing and agitation
Sleep	hypersomnia (greater than 8 hours per night)	hyposomnia (less than 6 hours per night)
Somatic complaints	much less than unipolar	much more than bipolar
Hostility	much less than unipolar; extreme passivity	hostile, irritable, and demanding
Clinical course		
Median age first onset	30 years old	43 years old
Median lifetime episodes	7 to 9	4 to 6
Prognosis	33% to 50% chronic impairment in intermorbid periods; 57% divorce rate; higher suicide rate than unipolars?	25% chronic impairment in intermorbid periods; 8% divorce rate; lower suicide rate than bipolars?
Genetic transmission		
Dominant X-linkage	implicated	not implicated
Polygenic	perhaps some forms?	implicated
Pharmacology		
Therapeutic		
Lithium	positive response in 80%	positive response in 40%
Tricyclics	?	perhaps better?
Prophylactic		
Lithium	positive for depression and mania	positive for depression
Tricyclics (one study)	positive for depression; negative for mania	positive for depression

Note. This table is necessarily simplified. See Depue and Monroe (in press-a) for a thorough review.

etiologic factors, biologic dysfunctions, and therapeutic response patterns. Hence, it is likely that some variables of the model will be appropriate for some forms of depression, while other variables of the model will be appropriate for other forms. Of course, it is always possible that some form of depression might "fit" all of the model's variables quite well and henceforth be conceived of as "helplessness" depression.

The necessity for greater precision in modeling with clinical depressions may be illustrated through a brief examination of Seligman's postulation of parallels between learned helplessness and reactive depression. Table 5 is adapted from a table presented in Seligman (1975, p. 106), of which Seligman argues, "As summarized in Table 5-1, the major symptoms of learned helplessness all have parallels in the symptoms of depression. This suggests that reactive depression, as well as learned helplessness, has its roots in the belief that valued outcomes are uncontrollable" (p. 105). While it is probable that precise analogies were not implied, Seligman has presented these commonalities as a means of promoting the potential of the learned helplessness model. Recently, these symptom similarities were cited in support of an extension of the model by others (Garber et al., in press). Therefore, discussion of some of these symptom parallels may serve to illustrate the complexities likely to arise in the future.

Passivity

In viewing passivity as a parallel between learned helplessness and reactive depression, Seligman (1975) states that "the lowered voluntary response initiation that defines learned helplessness is pervasive in depression. It produces passivity, psychomotor retardation, intellectual slowness, and social unresponsiveness; in extreme depression, it can produce stupor" (p. 84). There are several problems apparent in this parallel. First, passivity, psychomotor retardation, and stupor are not pervasive in depressive disorders. As may be

Table 5
Summary of Features Common to Learned Helplessness and Depression

Learned helplessness	Depression
Symptoms	
passivity	passivity
difficulty learning that responses produce relief	negative cognitive set
dissipates in time	time course
lack of aggression	introjected hostility
weight loss, appetite loss, social and sexual deficits	weight loss, appetite loss, social and sexual deficits
norepinephrine depletion	norepinephrine depletion
Cure	
electroconvulsive shock	electroconvulsive shock
norepinephrine stimulants	norepinephrine stimulants

Note. This table is adapted from Seligman (1975, p. 106).

seen in Table 4, these features characterize Bipolar 1 depression particularly, as measured by 24-hour behavioral ratings and 24-hour telemetric activity recordings; and in one study, similar characteristics were noted for Bipolar 2 cases (Depue & Monroe, in press-a). Indeed, some clinical reports have emphasized that bipolar depression is always characterized by passivity and psychomotor retardation. Unipolar 1 depression, on the other hand, is characterized much more by active pacing, agitation, irritability and anger, and somatic complaining (Depue & Monroe, in press-a). Moreover, psychomotor retardation has been consistently found to be the key feature of endogenous depression but not a feature of reactive depression (Fowles & Gersh, in press; Kendell, 1976; Mendels & Cochrane, 1968). Therefore, in terms of this parallel, bipolar depression would share the closest similarities to learned helplessness. And this raises the second problem: It is so-called reactive depression that is being modeled not bipolar depressive disorder.

The problem here may well arise from a third issue: the care with which symptoms must be defined. Seligman equates lowered response initiation with, among other features, psychomotor retardation and supports the equation by referring to the slower reaction time of depressives. Indeed, depressives are slower, but as Seligman points out, chronic schizophrenics are equally slow. The complication is that slowed reaction time may result from numerous factors, not the least of which might be poor concentration, also common in depressives. Hence, both Unipolar 1 and Bipolar 1 depressives might exhibit slowed reaction time due to the same factor (poor concentration), due to different factors (retardation in bipolar, poor concentration in unipolar), or due to some combination of these factors. Clearly, there are many ways in which a deficit may result. This raises the problem that measurement of certain well-specified clinical behaviors needs to be as specific as possible to avoid confounding by other variables.

In the depressive disorders, psychomotor retardation is not simply slowed reaction time. It is a symptom complex characterized behaviorally as delayed or slowed spontaneous and evoked purposeful motor activity, some rigidity of the musculature, often a hunched-over posture, unchanging facial expressions, and often a reduced frequency of blinking (Depue & Evans, in press). In its extreme forms, stupor may result. It is qualitatively similar in appearance to the akinesia seen in Parkinson's disease. Papeschi (1972) has suggested that this condition in depression may be related to functional deficiencies of dopamine in the extrapyramidal nigrostriatal pathway. Concordant with this hypothesis, some studies assessing the major metabolite of dopamine (homovanillac acid) in cerebrospinal fluid under probenecid administration have found reduced accumulations of the metabolite in retarded but not in agitated depressives, although conflicting results exist (see Depue & Evans, in press, for a review of this work).

This example also illustrates the importance of defining clinical behaviors precisely *and* the necessity of measuring these as specifically as possible. But when this is not done (or even when it is), suggestions that etiologic commonalities exist due to symptom parallels (as is present in the quote from Seligman above) must be offered most tentatively. This

would seem particularly true in the case where parallels are drawn between lowered response initiation found in human analogue studies of learned helplessness (e.g., longer response latencies) and behavioral psychomotor retardation found in clinical depression.

Time Course

Seligman (1975) sees a parallel in the fact that learned helplessness in animals follows a time course (dissipates in time) and so do depressive episodes. The analogy between time courses in animals who have been exposed to acute stress and time courses in clinical disease is crude at best. In addition, most diseases follow a time course, and so without further specification concerning the correspondence of time course parameters, this parallel is meaningless. Finally, strictly speaking, time course is not a symptom but one characteristic of the clinical course of a disorder.

Lack of Aggression

In discussing this parallel, Seligman (1975) states that "Depressed people are virtually drained of overt hostility toward others" (p. 89). This, of course, is a traditional concept derived from psychoanalytic notions concerning the defense mechanism of introjected hostility (as illustrated in the descriptors used in the parallel; see Table 5). As a general statement, the parallel is inadequate for these reasons: (a) Lack of hostility characterizes Bipolar 1 depressives but not Unipolar 1 cases (see Table 4) and (b) the parallel does not hold for all reactive/neurotic cases in view of Paykel's (1971) group of hostile depressives discussed above. Whether other neurotic cases are "drained" of overt hostility is an empirical question. Hence, currently this parallel is best applied to Bipolar 1 depression. Again, the need to make finer distinctions within the depressive disorders is evident.

Norepinephrine Depletion

In this case, a parallel is drawn between the absolute deficits in norepinephrine found in rats subjected to stressors (Seligman, 1975; Weiss, in press) and a hypothesis about catecholamine functioning in depressives. According to Seligman (1975), this hypothesis "claims that [norepinephrine] is depleted at appropriate sites in the central nervous system of depressives" (p. 91).

There are several shortcomings concerning this parallel. First, norepinephrine depletion is not typically viewed as a symptom of depression but as a hypothesized etiologic factor. Second, the parallel is an oversimplification of a complex issue. Strictly speaking, the catecholamine hypothesis does not claim that the catecholamine dysfunction in depression is simply one of depletion. Rather, it "proposes that some, if not all, depressions are associated with an absolute or relative deficiency of catecholamines, particularly norepinephrine, at functionally important adrenergic receptor sites in the brain" (Schildraut, 1965, p. 509). While this statement of the hypothesis is more intricate than the notion of norepinephrine depletion, it also has been criticized as too simplistic and inconsistent with the nature of biochemical processes and available data (Baldessarini, 1975).

Currently, the catecholamine hypothesis might best be viewed as suggesting that *some* depressions are *associated* with a *functional* deficiency of catecholamines at certain receptor sites in the brain, although this is probably an oversimplified statement (Baldessarini, 1975). The qualifier "some depressions" is important because Maas (1975) reviewed data suggesting that biochemical subtypes of depression may exist where functional norepinephrine dysfunction characterizes only one subgroup. The term "associated with" is important in that a claim of primary etiology is avoided.

The term "functional" means that no absolute deficiency may exist. It suggests that some of the behavioral manifestations and some of the biochemical and pharmacological data indicate that the central nervous system is functioning *as if* a deficiency in catecholamines existed. Such a state of affairs could arise due to dysfunction in any one of a number of critical aspects of catecholamine functioning, including synthesis, storage, release, sensitivity of the receptor, and re-up-

take, to mention just a few (Depue & Evans, in press).

In light of these complexities, to suggest that a parallel exists between norepinephrine depletion in rats exposed to acute stressors (a complex issue in itself) and hypothesized functional alterations in central catecholaminergic metabolism appears to us to be a gross oversimplication and unwarranted.

Conclusions

The few examples discussed above serve to illustrate the complexities involved in drawing parallels between learned helplessness and clinical depressions. Several themes that cut across these examples are apparent: (a) The heterogeneity inherent in the depressive disorders requires that the modeling process employ precise specification of depressive subtypes or at least specification of a clinical group's features; (b) some of the symptom parallels more adequately apply to Bipolar 1 depression (or at least some form of endogenous depression, which is true also of the electroconvulsive shock parallel) rather than reactive/neurotic depressions; (c) more precise definition and more specific measurement of symptoms or other variables (e.g., biochemical) used in analogies is required; and (d) suggested etiologic commonalities derived from symptom parallels will require proposed qualifications and greater tentativeness.

Problems in Defining and Identifying Depressed Subjects in Human Analogue Studies of Depression

The first section of this article indicates that depressive populations are characterized by extreme heterogeneity in their manifestations and, most probably, in etiologies. In recent years, a plethora of research has been aimed at deriving distinct types of depressive disorders (as represented by the unipolar–bipolar distinction) or at identifying dimensions that may clarify the underlying nature of the heterogeneity (as represented by some researchers' views of endogenous–reactive depressions). Less attention has been directed toward the mild end of the depressive spectrum, and currently, their natural history and clinical course are obscure.

It is unclear whether the mild depressive states occurring in relatively normal, nonclinical individuals are viewed best as representing the mild end of a dimension of severity, where clinical depressives occupy the opposite extreme position (the quantitative view), or as distinctly different forms of depression (the qualitative position). Proponents of the latter position argue that mild cases manifest mainly the mood component of depressive reactions and not the full range of features found in clinical depression, a disorder that becomes biologically autonomous and assumes the dimensions of illness, namely, disruption of psychomotor and vegetative functions, morbidity, mortality, autonomous course, prognosis, and response to pharmacological therapies (Akiskal, in press; Whybrow & Parlatore, 1973).

In light of these issues, the analogue studies of depression within the learned helplessness framework take on added significance. The assumption in this literature is that of the quantitative viewpoint: Mildly depressed college student subjects fall on a dimension of severity with clinical depressives (forgetting for the moment the subtype problem raised in the preceding section). Much, but not all (see Klein & Seligman, 1976, p. 25), of the basis for this position rests on the fact that (a) the mean score on the Beck Depression Inventory (BDI; Beck, 1967) for depressed college students (ranging approximately from 12–16) is *only slightly less* than the mean score for mildly depressed patients reported by Beck (1967) and Metcalfe and Goldman (1965), that is, 18.7 and 14.3, respectively,[4] and (b) high correlations are found between BDI scores and deficit measures, with BDI scores ranging *as high as* the reported means for

[4] Although a minor point, Beck's (1967) figure is difficult to interpret clearly because his sample was composed of both outpatients and inpatients of varying diagnoses, where depressive disorders only represented 37% of the cases. Therefore, this figure does not reflect the level of depression in depressed cases only but also the level of depression as a symptom in other disorders, as well as the contribution of general pathology of the other disorders to the total score.

severe depression (actual scores and percentage of subjects at this level not reported). These facts represented part of the argument employed by Klein and Seligman (1976) in countering the possibility that their depressed college students "are qualitatively different from depressed patients" (p. 25). They suggest that their depressed subjects are only quantitatively different and conclude: "So, generalizing our results to the psychopathology of depression seems reasonable" (p. 25).

It is a crucial point to note that these arguments hinge on one important implicit assumption: On the basis of their subject selection procedure (i.e., BDI scores), subjects can be defined and identified as *some* form of *depressives*. It is clear that this implicit assumption is operating, for arguments concerning whether one's depressed subjects are quantitatively or qualitatively related to other clinical forms of depression can only be meaningfully and *logically* entertained *after* it is certain that a depressed group has indeed been identified.[5] The BDI method of defining and identifying depressed subjects embodies many critical problems. These problems relate to issues concerning the use of rating scales as diagnostic instruments, in general, as well as to the specific problems inherent in self-rating scales (such as the BDI) as opposed to observer rating scales and clinical diagnostic procedures. We now turn to these problems.

In learned helplessness research, a criterion cutoff score of nine on the BDI, a figure that represents the mean BDI score for college students, is used to define college student subjects as depressed or not depressed.[6] While Klein and Seligman (1976) have stated that the terms "depressed" and "nondepressed" are just convenient labels, as we have pointed out, their quantitative (as opposed to qualitative) arguments with respect to their depressed subjects clearly indicate that these labels are being used with a more specific meaning.

The use of the BDI in this fashion would be the equivalent of its use in clinical research as a means of diagnosis (identification of members of a class). This is not an appropriate use of the BDI or, for that matter, any rating scale, be it a self-rating or observer rating scale. Such scales were developed originally for the purpose of estimating reliably and validly the *severity* of illness in individuals being examined (Carroll, Fielding, & Blashki, 1973). For instance, in discussing his observer rating scale (which is the most frequently used instrument in clinical research), Hamilton (1960) has always insisted that his scale was not to be used for diagnosis, and that it was meant only to give a measure of severity in patients *already diagnosed* on clinical grounds as suffering from a primary depressive disorder. Therefore, within a research framework, these scales have been used as a means of matching groups of subjects on severity of depression in or across centers or as a means of relating severity to dependent measures—not as a means of diagnosis. Even recent research comparing normal depressions to clinical depressions has used these scales only as supplementary data to diagnoses based on extensive information of clinical and social characteristics obtained in interviews (see Weissman, Prusoff, & Pincus, 1975, for a review and a study).

There are several reasons why rating scales, such as the BDI, are inadequate when used to identify depressed cases, and they all relate to the problem that an elevated score on these scales could result from a number of inde-

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[5] It is probably fair to say that we are overstating the case here to some extent. However, at the least, most articles on learned helplessness have not been at all clear concerning this issue, whereas others mix general caveats with claims that their findings may be reasonably generalized to clinical populations and to the testing of clinical treatment efficacy. While Klein and Seligman (1976) are clearer on this issue than others, the last two paragraphs of this article raise questions mainly concerning to which form of depression their depressed students may be related, and the qualitative–quantitative nature of this relation. What is not addressed is the issue of whether their subject selection procedures allow them to make *any* assumptions as to the qualitative or quantitative relation of their subjects to clinical depressives. Hence, as the model is extended to clinical populations, the issues we raise here and below will be of the utmost importance.

[6] In that a score of nine on the BDI represents the mean for college students, such a definition of depressed subjects implies an incidence of depression in the general population that is decidedly greater than most population surveys have found.

pendent factors. There are no symptoms that are unique to the depressive disorders. Hence, an elevated score could result for an individual who is relatively normal but unhappy, sad, or lonely at the moment (Hogarty & Katz, 1971; Katz, 1970; Weissman et al., 1975) or who is suffering from a loss of self-esteem (Zung, 1972) or loss of a loved object. Also, an elevated score may be obtained from individuals suffering from more chronic mild depressions who seek help from a general practitioner (Carroll et al., 1973) or from some other medical or psychiatric disorder (secondary depression; see Akiskal, in press) as well as from major primary depressive disorders (Carroll et al., 1973). In the absence of other sources of history, psychosocial, and clinical data, a score on a rating scale is virtually uninterpretable with respect to diagnostic concerns.

It is interesting to note that this use of a BDI score by learned helplessness researchers reflects a confusion similar to that discussed above concerning the use of a reaction time deficit as evidence for a parallel to psychomotor retardation (the lowered initiation of responses deficit). In both cases, there is failure to realize that such indicators do not always have a specific meaning but could result from a host of independent factors.

In any case, the above discussion points to a very general reason why rating scales are inadequate for diagnostic purposes: They are restricted to a limited range of accessible information. Scales only reflect information on the intensity of symptoms, whereas diagnoses are typically formulated on the basis of a wide range of information available to the clinician, including signs and symptoms, characteristics of onset, previous clinical course and behaviors, psychosocial characteristics, intermorbid adjustment level, and the presence or absence of other medical or psychiatric disorders. Moreover, rating scales seldom incorporate the full range or variations within categories of signs and symptoms available to the clinician (Carroll et al., 1973).[7]

Other problems associated with the BDI in respect to its diagnostic use are more specific to the self-rating format as opposed to the observer rating format. The problem of a restricted range of information arises again in that it is much more at issue with self-rating than observer rating scales. Whereas in observer rating scales (such as Hamilton's, 1960), the clinician completes his ratings taking into account information from all available sources (including a clinical interview) concerning the patient's behavior during the preceding week, self-ratings take into account only the patient's subjective estimates of his symptoms as they are experienced at that point in time. These latter ratings probably reflect a different dimension of depressive disorder than the clinician's ratings (Raskin, Schulterbrandt, Reatig, & McKeon, 1969). Beck (1967) was aware of this problem, and typically, BDI administration involves the reading of statements to the patient. The rater then evaluates the patient's response and scores each item. Thus, the BDI is actually an "interview-assisted" scale rather than a self-rating in the usual sense, although it has not been so used in learned helplessness studies.

Inherent in the above problem lies another difficulty with self-ratings: Patients as well as normals with mild depressive states do not have the clinical perspective of clinicians for rating the severity of their distressing symptoms. In addition, the meaning of items may be viewed from different perspectives by patients and normal depressives but be rated the same. For instance, "I blame myself for my faults" or "I hate myself" (alternatives to two different items on the BDI) both may be adopted as responses by patients and depressed normals, but it is quite possible that further elaboration on these items would reveal different perspectives.

This brings us headlong into the issue of sensitivity, that is, the ability of a rating scale to distinguish normal but mildly depressed individuals from those with an illness of psychotic proportions. All of the above factors will affect a scale's sensitivity, and self-rating scales may be particularly troublesome. For instance, Carroll et al. (1973) compared the relative sensitivity of observer ratings and

[7] For instance, the BDI does not incorporate items related to psychomotor or locomotor alterations.

self-ratings in patients ranging from severe to mild depression. The Hamilton Rating Scale (HRS; Hamilton, 1960) and the Zung Self-rating Scale (SRS; Zung, 1972) were administered before treatment to inpatient, day patient, and general practice patients carefully diagnosed as primary depressive cases. Whereas the HRS discriminated significantly between all three groups, the SRS did not differentiate significantly inpatients from day and general practice patients but did discriminate between day and general practice patients. An equivalent study comparing the HRS and BDI over a wide spectrum of depressions has yet to be done (see Carroll et al., 1973, for a review of this area).

The Carroll et al. (1973) study revealed one important factor that may interact with a scale's sensitivity and that is relevant to the use of the BDI, that of orientation, a scale's loading for subjective feelings or for behavioral and somatic features. Most currently used scales are overweighted toward subjective feelings, which are even less specific than behavioral and somatic features. Carroll et al. have suggested that sensitivity across the spectrum of depression might be increased if scales adopt a behavioral–somatic orientation. They found that the most powerful correlations between the HRS and the SRS in their study emerged for items with restricted meanings and an objective orientation (suicide, insomnia, work and interests, gut symptoms, and weight loss), while no correlations of significance were found between the objective and subjective ratings of agitation, somatic anxiety, and general somatic symptoms. The important implication of these findings for self-rating scales is that an objective behavioral–somatic orientation might avoid to some extent the problem that patients have in quantifying the degree of their symptoms.

There are also important implications here for the use of the BDI. The total behavioral plus somatic component of questions comprising the BDI is 33% (as opposed to 50% to 80% for the HRS, depending on how certain items are rated; Carroll et al., 1973). The heavy loading on subjective items on the BDI may create problems in its ability to differentiate between mild depressions in relatively normal subjects and more severely depressed clinical cases, since the more subjective mood and cognitive components of depression may be less discriminating than behavioral and somatic components for these populations (see discussion below). Were this the case, basing a quantitative view of depression in college students on BDI score levels—as has been done in the learned helplessness literature—would be a questionable procedure.

Recent research comparing normal individuals with depression and clinical depressed inpatients and outpatients bears directly on this issue (Hogarty & Katz, 1971; Katz, 1970; Weissman et al., 1975; Zung, 1972). With the exception of one study (Brauzer & Goldstein, 1973), the general conclusions arising from this research are that the behavioral, somatic anxiety, and other somatic components of depression are by far the best discriminators between clinical depressions and normal depression and unhappiness; patients are found to be markedly more impaired on the former. The central mood of depression generally does not differentiate, in that severity of depressed mood in normals can approach the level of outpatients. Moreover, subjective feelings of sadness, loneliness, and loss of self-esteem are quite pronounced or even somewhat more severe in normal depression. Thus, normal depressions appear to be best characterized by pronounced unhappiness, sadness, and loneliness. These symptom differences are interesting in that Weissman et al. (1975) found that in an interview, the normals attributed their problems to having recently moved to a new area, feeling dislocated, and lonely. Improvement, which was evident in the large majority of cases at a 4-month follow-up, was attributed by the normal subjects to finding a satisfying job or educational plan and receiving practical help.

These findings clearly indicate that in view of the BDI's heavy loading on the subjective feelings of depression, this instrument may be a poor discriminator (less sensitive) between normal individuals in a state of sadness, unhappiness, and loneliness and a moderately depressed clinical population. Until research bearing on this issue is undertaken, claims that depressed college students are qualita-

tively related but quantitatively less severe to clinical primary depressions must be viewed as unsupported.

Conclusions

We have attempted to illustrate the inadequacies inherent in defining and identifying normal depressed individuals by use of rating scales when claims to this group's relation to clinical populations are advanced. The problems in this practice arise from the inappropriate use of an instrument originally designed to estimate intensity of symptoms for actual diagnostic purposes. Special problems surrounding this issue accrue when a self-rating scale is used, including the following: (a) a greatly restricted range of information used in arriving at ratings, (b) a lack of clinical perspective on the part of individuals quantifying their distressing symptoms, (c) sensitivity to the spectrum of depression, and (d) especially with respect to the BDI, the relative loading on a subjective feelings orientation as opposed to a behavioral–somatic orientation. While all of these problems may limit the ability of the BDI to discriminate between normal and clinical depression, recent research comparing these two populations suggests that the BDI's heavy loading on subjective feelings may be most problematic. In view of these complexities, it cannot be assumed that the depressed college student subjects used in learned helplessness research have any qualitative relation to clinical depressions until more appropriate clinical data on this issue are gathered.

Concluding Remarks

The impressive progress that the learned helplessness model of depression has enjoyed augurs well for its potential significance in clarifying the nature of the depressive disorders. In the development of any model, numerous difficulties inevitably arise, and generally, creative responses to these difficulties enhance the model's applicability. With this process in mind, throughout this article we have focused on what we perceive to be significant issues that will require attention in extending the model to clinical populations.

The depressive disorders present the researcher with enormous complexities with respect to the heterogeneity of their clinical manifestations. No less troubling is the fact that this behavioral heterogeneity is equaled by diversity in familial, genetic, biologic, psychosocial, and probably, etiologic factors. Hence, simple dichotomies, such as the endogenous–reactive/neurotic distinction, and all-encompassing disease entities, such as Kraepelin's (1921) notion of manic–depressive illness, have proven to be inadequate conceptualizations in light of the growing body of clinical data. The first section of this article, then, represents an attempt to provide several clinical frameworks within which to extend the learned helplessness model to clinical depressions. As Seligman suggests, it is highly unlikely that learned helplessness serves as the primary etiologic factor in all forms of depressive disorders. The task for researchers is to determine which form best fits the model, and it is our hope that the discussion above promotes that effort. But, as would be in keeping with the multifactorial nature of etiologies in many clinical illnesses (Depue, in press-a), it is possible that learned helplessness will not serve as the sole etiologic factor in any form of the clinical depressions. If this be the case, a different form of theorizing will need to be developed in the learned helplessness literature than has been evident thus far.

Irrespective of the clinical form of the depressive disorders one is addressing, the process of constructing a model for a clinical group will require great care in the drawing of analogies. The second section of the article illustrates this through an examination of several parallels between learned helplessness and clinical depression put forth by Seligman (1975). The discussion noted the lack of clear-cut parallels between learned helplessness and reactive/neurotic depressions. At least two recommendations for future model construction were suggested: (a) more precise specification of depressive subtypes or a clinical group's features and (b) more precise definition and more specific measurement of symptoms or other variables used in analogies. The more general caveat was that suggested

etiologic commonalities derived from symptom parallels will require qualifications and greater tentativeness.

The last section of the article focused more specifically on the assumption that the depressed college student subjects employed in learned helplessness research are qualitatively related to but quantitatively less severe than clinical depressives. Because there are no appropriate data bearing directly on this issue, we chose to illustrate the problems inherent in such an assumption by examining the arguments used to support it, namely, elevated BDI scores and high correlations of these scores with deficit measures. This discussion brought into focus numerous problems in using the BDI or any rating scale for purposes of defining and identifying individuals thought to be qualitatively related to clinical primary depressive cases. These need not be reiterated here. The general thrust of the discussion illustrates the necessity of investigating the clinical nature of subjects used in learned helplessness research in the future.

One additional issue deserving attention is that the group of subjects scoring nine or above on the BDI has been treated as a homogeneous population. This may, indeed, not be the case. In view of the findings that normal depressions are characterized by pronounced sadness, loss of self-esteem, and loneliness, a heightened BDI score in this population may represent a nonspecific indicator of a variety of personal problems that result in a feeling of sadness and helplessness. Investigation into the nature of these various potential problem areas could serve as one additional extension of the learned helplessness model. The work of Weissman et al. (1975) serves as an excellent example for this type of research. They posed the interesting question of whether the sadness and impaired ability to cope in the face of stressful life circumstances found in their depressed normals might represent one mild "depressive" feature of individuals who *do not* show more severe forms of depression, but who *do* have a positive family history for affective disorders. Alternatively, and/or additionally, such individuals might be characterized by an expectancy of inability to control future outcomes or life stressors. Such issues raise exciting possibilities for the learned helplessness model of depression.

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